Chapter 4. Facial Nerve Paralysis Examination

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The facial nerve provides motor, sensory, and parasympathic innervation to the head and neck. The functional and aesthetic consequences of facial nerve paralysis can potentially be physically and psychologically devastating. Facial palsy is almost invariably accompanied by severe emotional distress. A complete history and thorough examination should be the primary focus of the treating physician. The goal of this chapter is to simplify the clinical evaluation of patients with facial nerve pathology. There are distinct aspects of the history, evaluation, and treatment strategy that differ between patients with acute and chronic paralysis. We separate patients with chronic facial paralysis into the following four categories: facial paresis without synkinesis, facial paresis with mild synkinesis, facial paresis moderate to severe synkinesis, and complete, flaccid facial paralysis.

History

The history for patients presenting with facial palsy serves to narrow the differential diagnosis (**Table 4.1**). In the evaluation of an acute event, the onset and grade of the facial palsy are key determining factors for the etiology, prognosis, and additional laboratory and diagnostic testing.

The priority during the evaluation of new onset facial paralysis without a clear cause (e.g. acoustic neuroma surgery, parotidectomy, trauma, etc.) centers on identifying the etiology of the paralysis and preventing ocular complications. All patients presenting with facial nerve paralysis should have the following documented:

1. Duration of paralysis

2. Onset: immediate versus progressive
3. Inciting factors: pregnancy, stress, etc.
4. History of previous facial nerve paralysis
5. Family history of facial nerve paralysis
6. Systemic medical conditions (autoimmune diseases, diabetes)
7. Skin changes or rashes around ear, face, neck, chest, or back
8. Prodromal symptoms such as nasal congestion, sore throat, fever or arthralgias
9. History of perioral herpes simplex virus infection
10. Travel history
11. Otologic symptoms including ear drainage, hearing loss, vertigo, otalgia, or aural fullness
12. Recent tick bite or camping
13. Full oncologic history including cutaneous neoplasms of the face
14. Recent trauma
15. Past surgeries including surgeries to the ear and central nervous system
16. Neurological symptoms including cranial neuropathies, weakness or tingling

In patients with long-standing facial nerve paralysis, it is equally important to obtain the history of initial paralysis as detailed above. In addition, the functional and aesthetic concerns associated with chronic facial nerve paralysis need to also be addressed. As aesthetic concerns are common in this population, it is important to document such information in the patients' own words. The possible sequelae of chronic facial nerve paralysis include:

1. Nasal airway obstruction

2. Oral incompetence
3. Aberrant facial nerve regeneration leading to involuntary or uncoordinated facial movements (synkinesis)
4. Buccal mucosa irritation
5. Visual changes, ocular irritation, lagopthalmous, dry eye syndrome, epiphora, corneal ulceration, blindness
6. Facial asymmetry due to flaccid atrophic muscles, loss of tone, synkinesis or volumetric loss
7. Loss of dynamic furrows
8. Brow ptosis and asymmetry
9. Effacement or deepening of the nasolabial folds
10. Drooping or elevation of the oral commissure
11. Dimpling of the chin (peau d'orange)

It is imperative that an underlying neoplasm is ruled out in all patients with facial nerve pathology, both acute and chronic. The clinical features that raise our suspicion for a neoplastic entity include:

1. Slowly progressive facial paralysis
2. Additional cranial neuropathies
3. History or presence of suspicious skin lesion or cutaneous malignancy
4. Parotid mass
5. Facial twitching
6. Absence of facial nerve recovery 4 months after onset of symptoms

7. Concurrent sensorineural hearing loss, aural fullness, tinnitus
8. Concurrent vestibular symptoms
9. Ipsilateral recurrence of facial paralysis
10. History of neoplasm

Any patient with the above features requires additional radiological imaging (MRI, CT) to rule out underlying malignancy.

Physical Examination

Head and Neck Examination

In patients presenting with facial nerve paralysis, a complete head and neck physical examination is warranted. Particular focus is placed on the ear, mastoid, parotid gland, facial skin, and cranial nerves.

The external ear is examined for evidence of an erythematous or vesicular rash suggestive of Ramsay-Hunt syndrome, a condition caused by varicella-zoster virus. The skin and scalp should also be inspected for scars that could indicate previous cutaneous neoplasm or traumatic injury.

The temporal bone and ear contents should be examined in a comprehensive fashion. The mastoid is palpated for tenderness that can be associated with fractures of the temporal bone or mastoiditis. Ecchymosis over the mastoid process following a traumatic event (Battle's sign) is suggestive of a basilar skull fracture. Multiple middle ear processes such as acute or chronic otitis media, middle ear effusion, hemotympanum, tympanic membrane perforation, neoplasm, and cholesteatoma can cause facial nerve paralysis. In patients suspected of having middle ear pathology, it is imperative to perform a complete microscopic examination of the region or refer the patient to a neuro-otologist. If

any abnormalities of the middle ear are appreciated or the patient subjectively complains of hearing loss, an audiogram is obtained.

The parotid gland should be palpated for masses or lesions that may impinge on the facial nerve. Sparing of individual branches of the facial nerve increases suspicion for a parotid neoplasm, however this finding may also be observed with iatrogenic injury. The neck is palpated for masses such as metastatic nodes or parapharyngeal tumors.

A complete neurological examination including vestibular and cranial nerve testing is essential. Additional neuropathies raise suspicion for tumors of the temporal bone, skull base, nasopharynx, or central nervous system.[1] Concurrent vestibulopathy in the setting of facial nerve paralysis is a poor prognostic sign. If clinical suspicion for a nasopharyngeal mass arises, fiberoptic examination of the nasopharynx is undertaken.

Facial Nerve Examination

The majority of patients with a peripheral facial nerve paralysis will demonstrate ipsilateral weakness of all muscles of facial expression, with only 2% of patients having bilateral involvement.[1] Central etiologies of facial paralysis spare the frontalis muscle and are relatively rare.

During the facial nerve examination, all patients are asked to perform a variety of movements including forehead elevation, eye closure, nasal wrinkling, whistling, pursing of lips, soft smile, full smile, and showing of all dentition (**Fig. 4.1a,b,c,d,e,f,g,h,i**). The muscles of the forehead including frontalis, procerus, corrugator supercilli, and depressor supercilli are assessed. The frontalis, a brow elevator, is innervated by the frontal branch of the facial nerve, while the brow depressors such as the procerus, corrugator supercilli, and depressor supercilli have dual innervation from the frontal and zygomatic branches. Position of the eyebrows should be assessed for symmetry as well as location in

relation to the supraorbital rim.

Periocular examination emphasizes the shape and function of the eye. Bell's phenomenon, or upward rotation of the eye on attempted eye closure, should be confirmed in all patients. Patients with facial paralysis that demonstrate poor Bell's phenomenon are at an exceedingly high risk of developing corneal ulceration and blindness if proper eye protection measures are not instituted (**Fig. 4.2a,b**). The height of the lateral canthus should be 2 mm superior to the medial canthal angle. The orbicularis oculi and the levator palpebrae are responsible for eyelid opening, closure, shape, support, and tear pumping. The orbicularis oculi receives dual innervation from the frontal and zygomatic branches. The levator palpebrae, an upper eyelid elevator, is innervated by the oculomotor cranial nerve and therefore not affected by facial nerve palsy. Normal palpebral fissure and ocular width is approximately 12 mm and 29 mm respectively. Patients with total facial paralysis have poor eye closure leading to lagophthalmos. On the other hand, patients with synkinesis often have narrowing of their palpebral fissure leading to asymmetric eyes.

The lower lid lash line should be positioned at the lower border of the lower limbus (**Fig. 4.3**). If it is inferiorly displaced, then the patient has lower lid malposition. Marginal Reflex Distance (MRD2) is the distance measured between the light reflex and central portion of the lower lid when a patient's eye is in the neutral position (**Fig. 4.4**). Lower lid malposition is present if the MRD2 is significantly greater than 5.5 mm. Lower eyelid tone and support should be verified by the snap and lid retraction test (**Fig. 4.5a,b**). If the lid does not snap back into its anatomical position in 1 second, the puncta displaces more than 3 mm, or the lid distracts more than 7 mm then lid laxity is diagnosed.

The midface region has significant arborization of the buccal and zygomaticus branches of the facial nerve and as a result, all muscles in this region likely have dual innervation. The buccal nerve

is the dominant branch to the buccinators and supplies part of the dual innervation to the orbicularis oris and the depressor angulii oris. The zygomatic nerve is the dominant branch to the orbicularis oris, zygomaticus major, zygomaticus minor, levator angulii oris, levator labii superiorus, and levator labii superiorus alaeque nasi (all lip elevators). The mentalis, depressor angulii oris, depressor labii inferioris, and platysma are innervated by the marginal mandibular branch. The cervical branch of the facial nerve innervates the platysma muscle (**Table 4.2**; **Fig. 4.6**). The marginal mandibular nerve is a terminal branch and as a result is less likely to recover from injury.

There are three types of smiles that can be appreciated: zygomatic, canine, and full denture (**Fig. 4.7**). Zygomatic smile is dominated by the zygomaticus major and is present in 67% or the population. The canine teeth and lower teeth are typically not visible. Canine smile usually involves the activation of zygomaticus muscles as well as levator labii alequae nasi and is present in 31% of the population. 2% of the population has a full denture smile where all upper and lower teeth are appreciated due to the activation of both elevators and depressors.[2] Knowing the characteristics of the smile will allow the physician to better understand what procedures are necessary to create a natural appearing smile.

Assessment of Muscles for Facial Expression

The naïve examiner may look at the face and simply notice the presence of the facial paralysis. The more experienced examiner will look at the face and examine each muscle group individually. Each muscle group is tested twice: the first time the movement is examined is to determine how well the individual the muscle group functions. The second time the muscle group is tested is to determine if any other muscle group has abnormal muscle movement. Additional abnormal movement may indicate the presence of synkinesis or hyperkinesis.

An overall assessment of the muscle function is determined and then individual groups are

tested starting superior to inferiorly. When examining the patient with a facial paralysis, the examiner should first observe the patient's facial movements when they are speaking. Next, the examiner will start with top of the face and notice how the patient moves the forehead. The patient is asked to raise his or her eyebrow to assess the action of the frontalis muscle. The eyebrows should elevate similarly and equally on both sides. There should be symmetric furrowing of the forehead. Abnormal movement.of the obicularis oculi muscle is next tested by asking the patient close their eye. The patient is asked to close their eyes as tightly as possible. The patient is then asked to close their eye gently to notice how well the eye closes under less tension. The purpose of eye closure testing is twofold: assestment of orbicularis oculi function and also to determine how well the cornea is protected. When poor function of eye closure is present, the examiner should observe for a Bell's phenomenon: described as protective movement of the globe superior and lateral to protect the cornea under the upper lid when the lid cannot close. The amount of corneal exposure should be noted.

Next, the zygomaticus major muscle is tested by asking the patient to smile. A broad open mouth smile is first to tested followed by a gentle closed mouth smile. This demonstrates if the patient has significant movement and can also assesses for abnormal muscle movements such as synkinesis. The patient is asked to move their nose or wiggled their nose testing nasalis muscle movement. This can also assess the effect the paralysis is having on the nasal valve area. loss of this function can cause the patient to complain of nasal obstruction. Testing of the mouth area can assess any difficulty the patient may have with eating and articulation. The patient is asked to pucker and also to press their lips firmly together. Pressing the lips together is an excellent test of syskinetic muscle movement. Chronic facial paralysis patients that develop syskinesis will complain of abnormal movement and pressing of the lips together is the best test of oral-ocular syskinesis. The eye will close or narrow with lip tightening when

syskinesis is present. The patient is asked to press the jaw forward and tighten their neck muscles. This assesses the platysma's movement. This can be important when long-standing facial paralysis is present as abnormal bands of platysma tightening may be seen. After complete assessment of the facial nerve function, a facial nerve grade may be given to the patient; see chapter 5 for grading scales (**Fig. 4.8a-d; Fig. 4.9; Fig. 4.10; Fig. 4.11**).

Occasionally, it is helpful to have the patient videotape their facial nerve function for documentation purposes. This may be helpful if the patient lives a distance from the treating physician. A videotaping procedure is included as an appendix at the end of this chapter.

Isolated Facial Nerve Paralysis

Patients may present with isolated paralysis of one or more branches of the facial nerve. This is most commonly secondary due to trauma or iatrogenic injury. Patients with a frontal nerve paralysis will present with brow ptosis, loss of dynamic rhytids of the forehead and crow's feet. Lagophthalmos is rarely seen with an isolated frontal branch palsy. With loss of the zygomatic branch, patients may have difficulty closing the eye, effacement of the nasolabial angle, and decreased elevation of the lip with animation. Buccal branch injuries are subtle in nature. Patients will have a diminished ability to depress the lower lip and may complain of biting their buccal mucosa owing to denervation to the buccinator. In marginal mandibular nerve injury, patients have reduced capability to depress the lip during smile.

Congenital unilateral lower lip paralysis, CULLP, is secondary to agenesis of the depressor anguli oris so it is not a true disorder of the facial nerve.[5,6] Its clinical picture is similar to a marginal mandibular nerve injury (**Fig. 4.12**).

Isolated paralysis of the cervical branch has little sequelae and patients rarely complain of functional deficits.

Bilateral Paralysis

A subset of patients may have bilateral paralysis and either side may be categorized into the above functional categories. These patients most commonly have Möbius syndrome or bilateral Bell's palsy. Patients with complete bilateral paralysis have significant functional deficits including oral incompetence and speech impediments. The adverse psychological effects secondary to the inability to demonstrate facial expression are also significant. Frequently patients feel isolated and struggle to effectively communicate.

Differential Diagnosis

Facial paralysis is uncommon, with an estimated 30 cases per 100,000 each year. There is a wide ranging differential diagnosis for facial paralysis; the common etiologies are outlined in **Table 4.1**. Bell's palsy (idiopathic facial nerve paralysis) is the most common diagnosis, accounting for up to 70% of cases of unilateral facial paralysis.[7] Bell's palsy is a diagnosis of exclusion and all other potential etiologies should be ruled out prior to making this diagnosis.

Trauma is the second leading cause of facial nerve paralysis followed by Ramsay Hunt syndrome and neoplasm.[8] Facial paralysis in a patient with history of a previous cancer, particularly skin cancer, should be attributed to metastatic disease until proven otherwise.

Congenital etiologies include syndromes and teratogens, both of which are commonly associated with a wide variety of other congenital anomalies in addition to facial palsy. Orobello believes congenital facial nerve paralysis is an error in embryogenesis, not fetogenesis, and should be appropriately termed developmental facial nerve paralysis.[9]

Möbius syndrome, a rare congenital disorder of unclear etiology, is predominately characterized

by unilateral or bilateral facial nerve paralysis and abducens nerve palsy; however, involvement of other cranial nerves has also been reported. This disorder is associated with a variety of limb, orofacial, and chest-wall abnormalities.[10]

Roughly 30% of patients with ipsilateral recurrence of facial palsy were found to have tumors of the facial nerve or parotid gland, therefore, recurrent facial paralysis warrants diagnostic tests and imaging.[11] In cases of adult-onset bilateral facial nerve palsy, entities such as brainstem tumors, intracranial infection, Guillain-Barre syndrome, and Lyme disease are most common.[1]

Special Testing

Audiometry

Pure tone and speech audiometry should be considered in patients with facial nerve paralysis. This allows for documentation of hearing and identifies patients that may have simultaneous involvement of the eighth cranial nerve. Further, it establishes baseline hearing in cases requiring surgical or non-surgical intervention.

Topognostic Testing

Topognostic testing has historically been used in an effort to identify the exact site of a lesion. Theoretically, facial nerve branches proximal to the lesion should respond normally. Topognostic testing in localizing facial nerve lesions remains unreliable due to the variable anatomy of the facial nerve and its branches, the fact that lesions often fail to localize to one site of the nerve, and the variable recovery rates of differing neural segments.[8] The tear, salivary flow, and taste tests are not reliable predictors of outcome in cases of Bell's palsy.[12,13] Because of difficulty in obtaining accurate results, lack of prognostic applications, and the emergence of improved imaging techniques, topognostic testing is only

useful to supplement other diagnostic information and has a restricted role in current practice.

Shirmer's Test

Shirmer's test evaluates the function of the greater superficial petrosal nerve which supplies secretory fibers to the lacrimal gland. The greater superficial petrosal nerve branches from the facial nerve at the geniculate ganglion. This test involves placing sterile paper strips in the conjunctival fornix to stimulate tear production. Tear production after 5 minutes is measured and this value is compared between the eyes. A 25% decrease in tearing or less than 25 mm of lacrimation on the pathological side is an abnormal test. An abnormal test would suggest a lesion proximal to the geniculate ganglion.

The Stapedial Reflex

The stapedial reflex tests the integrity of the stapedial nerve, a branch arising from the mastoid segment of facial nerve innervating the stapedial muscle. This bilateral reflex is elicited by either ipsilateral or contralateral acoustic stimulation. Responses are measured through alterations in acoustic immitance. A 50% decrease in the amplitude of the reflex is considered abnormal, signifying a lesion proximal to the stapedial nerve. The prognostic value of stapedial testing in acute facial paralysis has been studied. In a small series, all patients who had a normal stapedial reflex within 2 weeks of facial paralysis completely recovered in 12 weeks.[14] Conversely, an abnormal reflex is common in the first 2 weeks after facial paralysis limiting its prognostic role in predicting poor outcomes.[8]

Taste Tasting

Chorda tympani arises just proximal to the point at which facial nerve exits the skull base through the stylomastoid foramen. Chorda tympani passes through the middle ear and petrotympanic fissure to join the lingual nerve. It carries taste fibers to the anterior two thirds of the tongue and secretory fibers of the submandibular and sublingual glands. Taste testing involves application of stimuli to different sites

on the tongue and qualitatively compares responses. Taste testing is of no prognostic value in the acute phase of Bell's palsy as the majority of patients will have abnormal responses.[15]

Salivary Flow Test

The salivary flow test measures the secretion rates of the submandibular and sublingual glands. This technique requires cannulation of Wharton's ducts and saliva collected is compared between sides. Reduced flow suggests a lesion proximal to chorda tympani branching. Salivary flow testing can be both uncomfortable for the patient and time consuming. Further, it has little prognostic value and is rarely used.[8]

Electrical Testing of the Face

Nerve Excitability Test

The use of the nerve excitability test (NET) in the evaluation of facial nerve paralysis was first described in 1962.[16,17] This technique became popularized when Hilger introduced a nerve stimulator that was compact, inexpensive, and easy to use in 1963.[18] NET requires placement of a stimulating electrode over the facial nerve trunk or a peripheral branch. During a minimal excitability test, low energy pulsed current is steadily increased to the normal facial nerve until facial muscle twitching is observed. The threshold of excitation is defined as the lowest current producing a visible twitch. This process in then repeated on the paralyzed side and the threshold difference is calculated.

NET is particularly useful in differentiating physiologic blockage, or neuropraxia, from axonal degeneration.[16] With neuropraxia, electrical stimulation distal to the site of conduction block will produce a propagated action potential and subsequent muscle twitch. In these cases, NET will not demonstrate differences in threshold potential between healthy and paralyzed sides at any time point

after symptom onset. Conversely, in patients with more severe injuries ranging from partial axonal degeneration to complete axonal transection, NET can provide valuable information. Nerve excitability will remain normal until distal axonal degeneration occurs. However, this can take up to 3 to 4 days even after complete transection. Thus the usefulness of NET in the first days after pathological insult is limited.[19,20]

Variability exists amongst authors as to a significant threshold difference between paretic and normal facial nerves.[21,22,23] Laumens et al proposed that a threshold difference greater than 3.5 mA is a reliable sign of nerve degeneration and accurate predictor of poor prognosis.[17] Devi et al followed patients with facial palsy for six months with serial minimal excitability tests. Patients with a NET of greater than 5 mA had poor recovery unless the difference significantly improved within one week of symptom onset.[24]

The differences in the stimulation thresholds obtained in the NET may assist in predicting severity of injury and probability of recovery. The test relies, however, on subjective scoring and the standard value of significant threshold difference may differ between institutions making generalized guidelines difficult to describe. Due to the development of newer, objective electrophysiologic testing methods, the applicability of NET is restricted in current practice but useful in select clinical scenarios.

Maximal Stimulation Test

The maximal stimulation test (MST) is a modification of the NET that employs similar neurostimulators, electrodes, and electrode placement. While the NET measures the minimum current necessary to elicit a facial twitch, MST uses a level of current (maximal stimulus) at which the greatest amplitude of facial movement is observed. The maximal stimulus provides sufficient electricity to depolarize all axons. Current levels greater than maximal stimulus, or supramaximal stimulus, can be used as well. The

movements of the facial muscles on the paralyzed side are subjectively described in comparison to the healthy side as follows: equal, slightly decreased, markedly decreased, or absent when compared to the healthy side.[25]

In a study evaluating the prognositc application of MST in patients with idiopathic complete facial paralysis, patients demonstrating equal MST in the affected and paretic sides had a 92% chance of complete recovery.[26] An additional study supported this finding and found that complete recovery occurred in 3 to 6 weeks.[18] Conversely, in patients with markedly decreased or absent responses on the affected side, there was an 86% chance of incomplete recovery of facial function.[26]

The MST can be a useful test in the evaluation of a facial paralysis patient but it has similar limitations to the NET. It is a qualitative method relying on subjective observations and testing can be limited by pain experienced by the patient. As with NET, MST will be normal until the onset of Wallerian degeneration, which can take up to 4 days. MST has been shown to become abnormal earlier than NET, suggesting it may be a superior test.[25]

Electroneuronography

Electroneuronography (EnoG), or evoked electromyography, measures muscle action potentials elicited by supramaximal stimulation of the facial nerve. First described by Esslen and Fisch, this method involves placement of a bipolar stimulating electrode at the stylomastoid foramen and a second recording electrode in the nasolabial groove.[27,28] The exact placement of the second electrode has been challenged in recent studies, however the most appropriate muscle is likely the nasalis.[29] Supramaximal electrical stimulation is applied and the amplitude and latency of the elicited compound muscle action potential (CMAP) is recorded. The amplitude of the maximum response is compared between the affected and normal facial nerve. Expressed as a percentage, this value theoretically reflects the extent

of facial musculature denervation and correlates to the number of degenerated motor nerve fibers. This information can be used to objectively assess the amount of neural degeneration.[30] Therefore, if the amplitude of the response on the paralyzed side is 30% of the response elicited on the healthy side, approximately 70% of the motor axons on the injured side have degenerated.

The role for EnoG in cases of partial paralysis is limited because a full, spontaneous recovery can generally be expected in these patients. However, its use as a prognostic tool in complete facial nerve paralysis has been well studied and advocated by many experts.[15,26,28,31,32,33] Given that neural Wallerian degeneration does not occur until 3 to 4 days after the pathological event, EnoG testing before this time will demonstrate normal muscles responses, and therefore not be of any practical value.[32,34] Most proponents of EnoG support daily or every other day serial examinations until reductions in amplitude on the paralyzed side ceases. Once this steady state is reached, the maximum amount of nerve degeneration is determined. EnoG has limited clinical value after this point because clinical improvement will almost always manifest prior to electrical changes seen on an EnoG.

Most data predicts complete recovery when the paralyzed side shows less than a 30% reduction in amplitude of CMAP. For reductions in CMAP amplitude between 70 and 90%, full recovery can take from 2 to 8 months and mild to moderate residual deficits can be expected.[31]

Many authors have shown that CMAP amplitude reduction, or nerve degeneration, greater than 90% correlates with a poor recovery (**Fig. 4.13**).[26,31,32] Residual function will be moderately to severely limited and maximum recovery will be delayed between 6 to12 months.[31] Conversely, a prospective multicenter trial demonstrated that patients who did not reach 90% nerve degeneration on EnoG within 14 days of symptom onset returned to House-Brackmann grade I or II by 7 months.[33]

The reliability of EnoG in identifying candidates who will benefit from surgical intervention

has been studied.[28,33] Fisch established criteria for surgical decompression after neural degeneration of greater than 90% observed within 3 weeks of symptom onset. Using these guidelines, patients who underwent decompression had more satisfactory return of facial function than patients in the non-surgical group.[28] Gantz et al studied the value of surgery in Bell's palsy patients that had greater than 90% reduction in CMAP amplitudes and no voluntary motor unit potentials on EMG. Decompression within two weeks of symptom onset was associated with a 91% chance of returning to House-Brackmann grade I or II by 7 months. Patients who were treated with steroids only had a 42% chance of achieving a similar functional outcome. No benefit to surgical decompression after 2 weeks of symptom onset was demonstrated.[33]

May et al. studied the value of surgical decompression in Bell's palsy patients demonstrating greater than 75% reduction in Shirmer's test, salivary flow test, MST, and EnoG. Patients meeting these criteria underwent decompression of the facial nerve. Contrary to prior results published by Fisch and Gantz et al, no benefit of surgical decompression was demonstrated.[35] It is important to note that different types of surgical decompression were performed in the various studies. Fisch and Gantz et al decompressed the meatal foramen, labyrinthine segment, geniculate ganglion, and tympanic segment of the facial nerve while May at al did not decompress the meatal foramen.

While the role of surgery in Bell's palsy remains controversial, critics of EnoG cite poor test accuracy in predicting unfavorable outcomes. In a series of 23 patients meeting the surgical criteria originally proposed by Fisch, 80% were shown to have moderate to complete return of facial function.[36] EnoG is also susceptible to test-retest variability evidenced by amplitude ratios that are not constant with repeat measurements on the same subject.[37] However, there are reports of test-retest variability as low as 6.2 percent.[38] Taking an average of multiple tests performed during a single examination and

stimulating the nerve 10 to 20 times before recording amplitude has been shown to improve the accuracy of measurements.[39]

EnoG is currently the best available electrodiagnositc technique available for evaluation acute facial nerve palsy in the immediate postoperative setting.[13,22,26,40-43] In cases of acute onset complete paralysis, it provides physicians with vital information concerning the degree and rate of nerve damage. Most agree that valuable prognostic information can be extracted from EnoG data; however, no consensus concerning the use of EnoG in selecting patients for surgical decompression exists.

Electromyography

Facial nerve electromyography (EMG) involves placement of bipolar needle electrodes in the facial musculature that record electrical action potentials generated by spontaneous and voluntary muscle contraction. In contrast to other neurophysiologic tests, this test does not require active stimulation of the facial nerve and is the only test that can document reinnervation of the facial nerve.

During an injury to the facial nerve, there will be a decrease in the number of voluntary motor units that are innervated. EMG measures the voluntary firing of motor units of the facial musculature. In the acute phase of facial nerve paralysis, the continued presence of voluntary motor units 72 hours after symptom onset suggests that some motor axons remain intact, although the degree of injury cannot be assessed. Lack of voluntary motor units at this time confers a poor prognosis to complete recovery.[26] Motor units that fail to become reinnervated develop unstable resting membrane potentials, and will begin to spontaneous depolarize in positive sharp waves and fibrillation potentials (**Fig. 4.14**). These may develop between 10-21 days after the injury.[44,45,46] Two studies evaluated patients that displayed fibrillation potentials at 10-14 days after facial paresis and reported 80.8% and 86% positive predictive values of incomplete recovery.[44,46]

EMG is a vital tool in attempting to prognosticate outcomes for patients with complete nerve palsy and greater than 90% degeneration with EnoG. In some cases, greater than 90% degeneration recording on EnoG may be due to dyssynchronous discharges of the neurapraxic fibers. This haphazard firing prevents adequate summation of the myogenic action potential resulting in a significantly reduced or absent CMAP suggesting severe nerve degeneration.[33] In this scenario, the value of EMG in conjunction with the EnoG cannot be underestimated as it is imperative to identify false-positive EnoG. It has been demonstrated that despite markedly decreased EnoG (>90%), the prognosis for recovery is excellent if voluntary motor potentials are present on EMG.[15,32] Fisch has termed this the "early de-blocking" phenomenon. He attributes the presence or return of voluntary motor units to early reversal of the physiological conduction block responsible for the neuropraxia.[32] These patients would not need surgical decompression.

In patients with acute facial nerve palsy and no clinical resolution of symptoms 2-3 weeks after onset of paralysis, EMG is particularly useful. At this time point, many neurophysiological testing are no longer useful as the facial nerve may have lost excitability. The appearance of small, rapid, polyphasic potentials on EMG is an indicator of nerve regeneration and suggests that further functional recovery is still to come (**Fig. 4.15**).[21,27] The return of voluntary motor unit firing often precedes clinical recovery of the facial nerve. Serial EMG is the only test that can follow the degree and pace of facial nerve recovery and reinnervation.

EMG is complementary to other neurophysiologic tests in patients with facial paralysis. It is the only test to document and follow recovery of the facial nerve. It is invaluable in situations where greater than 90% degeneration occurs on EnoG to identify patients that have a good prognosis for recovery.

Blink Reflex

The afferent and efferent limbs of the blink reflex are mediated by the trigeminal nerve and facial nerve respectively. The reflex can be routinely elicited in healthy patients by either mechanical or electrical stimulation of the supraorbital nerve.[47] Resultant orbicularis oculi muscle consist of an oligosynaptic early response ipsilaterally (R1), and polysynaptic late responses bilaterally (R2, R2').[48,49] Abnormal blink reflex responses are characterized by differences in amplitude or latency of R1, R2, or R2' responses between affected and unaffected sides. In contrast to direct facial nerve testing, blink reflex testing provides information on the neurophysiologic status of the trigeminal nerve, pons, and the proximal intracranial segments of the facial nerve. The previously mentioned direct electrophysiologic facial nerve stimulation techniques cannot evaluate these neural pathways.[21,50]

Patients with facial nerve paralysis are expected to have amplitude reductions, increased latencies, or absence of ipsilateral R1 and R2 responses on the paretic side.[48,50] No significant difference in median value of amplitude or latency of the contralateral R2' component has been demonstrated when comparing a paralyzed facial nerve to controls.[50] In a study of 32 patients with Bell's palsy, increased R1 latency was the most common change occurring in 34.4% of patients.[51] Few studies have examined the utility of blink reflex as a prognostic measurement in peripheral facial nerve injuries. Kimura et al suggested that a return of the R1 reflex after one week was a good prognostic sign.[52]

Blink reflex abnormalities in patients with acoustic neuroma has also been described. This technique was first used as an adjunct in the diagnosis of cerebellopontine tumors however has been largely replaced by more sensitive and specific radiographic studies.[53]

The blink reflex provides valuable information concerning neurophysiologic status of trigemino-facial connections and function of the intracranial facial nerve.

Electrophysiologic Features of Residual Nerve Deficits

Synkinesis is common after a facial nerve insult. Clinically, synkinesis presents as involuntary contraction of one facial muscle upon voluntary contraction of a separate muscle. Various mechanisms have been proposed to explain the development of clinical synkinesis including peripheral ephaptic transmission, aberrant regeneration of facial nerve fibers, and hyperexcitability of the facial nucleus.[54] While aberrant nerve regeneration is the most widely accepted theory, no consensus has been reached.

Synkinetic Spread

Synkinetic spread can be quantitatively evaluated with blink reflex testing. Stimulation of the supraorbital nerve normally elicits a response only in the orbicularis oculi. Abnormal wave responses are recorded simultaneously from both the ipsilateral orbicularis oculi and orbicularis oris muscles in patients with clinical evidence of synkinesis. In a heterogeneous study population of 29 patients, Kimura et al described increased latency and decreased amplitude for R1 and R2 in both muscle groups. Comparison of reflex responses between the two muscle groups did not reveal any significant differences.[52]

Lateral Spread

Lateral spread, or ephaptic spread, can be evaluated electrophysiologically. It is characterized by stimulation of one facial nerve branch resulting in a late response of a facial muscle not normally innervated by that branch.[55,56] Formation of an artificial synapse at the site of injury and crossing over of impulses from one nerve fiber to another is the proposed mechanism.

Though synkinetic and lateral spread can be demonstrated electrophysiologically as described above, these responses appear after the development of clinical synkinesis in almost all cases. Therefore, they are not used to predict the development of synkinesis.[57] Objective measurements can be helpful

though in distinguishing volitional movement from synkinetic movement, especially in cases of subclinical synkinesis.

Blood Testing

In patients with facial nerve palsy, no single laboratory study exists that definitively confirms the diagnosis. Many studies have examined the prevalence of antibodies to herpes simplex virus (HSV) and varicella-zoster virus (VZV).[7,58-64] Studies have demonstrated that patients with Bell's palsy had significantly higher levels of IgM antibodies to VSV and IgM and IgG antibodies to HSV than controls.[58,60,64] Additional serologic studies may be considered to suggest or rule out other potential causes. These should be dictated by the clinical history and setting. Though non-specific, erythrocyte sedimentation rate and white blood cell count can help differentiate between infectious from non-infectious processes. When history involves tick bites or travel to endemic areas of Lyme disease, Lyme titers are indicated. HIV testing is indicated if signs of immunocompromise are noted or the patient has a history of intravenous drug use. Serum angiotensin levels may also be considered if sarcoidosis is suspected.

Conclusion

Facial neuropathy has wide ranging effects both in form and function of the head and neck. A comprehensive and detailed history and examination is fundamental in the evaluation and treatment of patients with facial neuropathy. Recent advances in electrophysiologic technology have greatly improved our ability to accurately prognosticate outcomes and guide further treatment. Interpreting electrophysiologic results in the context of history, physical examination, laboratory values, and radiographic findings will continue to improve patient care and ultimately clinical outcomes.

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Figure Legends

Fig. 4.1 Patient with Type A facial nerve function demonstrating normal facial movement. (**a**) At rest, the face is symmetric without discrepancy in the position of the oral commissure or lower lid position (**b**) During raising of the brows, the brow raises to similar heights and dynamic furrows from the frontalis can be appreciated (**c**) Eye closure is complete (**d**) During contraction of the nose, the buccal and zygomatic branches are evaluated (**e, f**) The patient is asked to make a soft and full smile (**g, h**) The competency of the oral cavity is tested by having the patient make "fish lips" and "puff out" their cheeks (**i**) The marginal mandibular branch is best tested by asking the patient to pull down their lower lip

Fig. 4.2 Normal and Abnormal Bell's Phenomenon (**a**) During normal eye closure, the Bell's phenomenon stimulates the eye to rotate superiorly to protect the cornea (**b**) In patients with abnormal Bell's Phenomenon, the eye does not rotate superiorly during attempted eye closure, leaving the cornea exposed and at risk for injury

Fig. 4.3 Position of the lower lid lash line

Fig. 4.4 Marginal reflex distance (MRD) is used in the evaluation of upper eyelid ptosis and lower lid retraction. Eyelid ptosis can be quantitatively determined by measuring the margin reflex distance-1 (MRD_1) A) The MRD_1 is determined by measuring the distance between the corneal reflex and the upper lid

Fig. 4.5 Snap and lid retraction test is used to test for lid laxity. The lid is distracted away from the globe. (**a**) If the puncta displaces more than 3mm from the medial canthal tendon or does not snap back into place in less than 1 sec then lid laxity is present (**b**) The lid is lax if it can be distracted more than 7-10mm from the globe

Fig. 4.6 Facial Musculature

Fig. 4.7 Classification of Smiles. (**a**) Zygomatic smile is dominated by the zygomaticus major and is the most common type of smile mechanism. The canine teeth and lower teeth are typically not visible. (**b**) Canine smile usually involves the activation of zygomaticus muscles as well as levator labii alequae nasi and is present in approximately one third of the population. (**c**) Full denture smile involves activation of both elevators and depressors. All the upper and lower teeth can be appreciated.

Fig. 4.8 Patient with mild facial paralysis. (HB grade 2) (**a**) At rest there is general symmetry and similar volumes of each side of the face. (**b, c, d**) During animation, the face becomes asymmetric while the paretic side displays minimal movement

Fig. 4.9 Patient with moderate facial paralysis. (HB grade 3) (**a**) At rest minor asymmetries can be noticed including depression of the oral commisure and elevation of the brow. (**b**) During brow elevation, minimal movement of the paretic face is observed. (**c**) During smile, there is moderate synkinesis with dimpling of the chin and moderate narrowing of the palpebral fissure. (**d**) Showing of the lower teeth produces minimal movement of the paretic side.

Fig. 4.10 Patient with moderate facial paralysis. (HB grade 4) (**a**) Patient with right sided partial facial paralysis without synkinesis. Asymmetry at rest including lower lid malposition and drooping of the corner of the mouth. (**b**) On gentile smile, the pulling of the risorus can be appreciated (**c**) On full smile, the asymmetry of the eyes is exaggerated and the smile appears "pulled" on the right paretic side (**d**) Patient has good movement of the marginal mandibular nerve and little synkinesis. (**e**) Volume loss is appreciated on the paretic side

Fig. 4.11 Patient with severe facial paralysis. HB grade 5 (**a**) At rest, a complete right flaccid facial paralysis produces volumetric loss, effacement of the nasolabial fold, descent of oral commissure, and severe asymmetry. (**b**) Patient raising the brows. (**c**) Substantial asymmetry and absence elevation of the oral commissure during smile. (**d**) Difficulty pursing the lips. (**e**) Right orbit displaying lagophthalmus, lower lid laxity, lower lid malposition, and ocular irritation

Fig. 4.12 Congenital unilateral lower lip palsy

Fig. 4.13 ENoG demonstrating greater than 90% reduction in compound muscle action potential in the paralyzed left facial nerve when compared to the contralateral side

Fig. 4.14 Positive waves and fibrillation potentials are signs of deinnervation

Fig. 4.15 Polyphasic motor unit potential is an indication of active reinnervation

Table 4.1 Common etiologies of facial nerve paralysis

Congenital	• Mobius Syndrome • Congenital Unilateral Lower Lip Paralysis • Hemifacial Microsomia • Melkersson-Rosenthal syndrome • Goldenhar Syndrome
Neoplastic	• Temporal Bone Tumor • Meningioma • Facial Neuroma • Acoustic Neuroma • Parotid Tumor • Cholesteatoma • Metastatic Disease
Traumatic	• Birth Trauma • Temporal Bone Fracture • Facial Laceration • Penetrating Trauma • Iatrogenic

Infectious	• Lyme Disease • Herpes Simplex Virus • Varicella-Zoster Virus • Cytomegalo-Virus • Hepatitis B • Hepatitis C • Epstein-Barr Virus • Mumps • Rubella • Tuberculosis • Acute/Chronic Otitis Media • Mastoiditis • HIV • Syphilis • Petrositis
Neurologic	• Guillain-Barre • Myotonic Dystrophy • Stroke • Multiple Sclerosis
Idiopathic	• Bell's Palsy • Recurrent facial palsy
Systemic	• Sarcoidosis
Metabolic	• Diabetes Mellitus • Osteopetrosis

Table 4.2 Innervation of the Muscles of Facial Expression

Facial Nerve Branch	Muscle Innervated	Muscle Origin	Muscle Insertion
Temporal	Frontalis	Galea aponeurotica	Skin above the eyebrows
	Procerus	Fascia of the nasal bone and upper nasal cartilage	Skin in center of the forehead between eyebrows
	Corrugator Supercilli	Orbital rim near the medial canthus	Deep surface of the frontalis muscle
	Orbicularis Oculi	Medial palpebral ligament, lacrimal crest, or from bone on the medial orbital wall	Circumferentially around the orbit
Zygomatic	Procerus	*as above*	
	Corrugator Supercilli	*as above*	
	Orbicularis Oculi	*as above*	
	Zygomaticus Major	Zygomatic bone	Orbicularis oris near angle of the mouth

Buccal	Zygomaticus Major	*as above*	
	Zygomaticus Minor	Zygomatic bone	Orbicularis oris near angle of the mouth
	Levator labii superioris	Maxilla just above the infraorbital foramen	Upper lip
	Levator labii superioris alaeque nasi	Frontal process of the maxilla	Ala of the nose and upper lip
	Risorious	Fascia of masseter below zygomatic arch	Corner of the mouth
	Levator Anguli Oris	Maxilla inferior to the infraorbital foramen	Corner of the mouth
	Nasalis	1. Transverse portion: upper jaw near the canine tooth 2. Alar portion: upper jaw and nasal cartilage	1. Transverse portion: nasal cartilage on the bridge of the nose 2. Alar portion: skin of nostril
	Depressor Anguli Oris	Mandible near the attachment of the platysma	Corner of the mouth
	Depressor labii inferioris	Mandible	Skin and muscles of the lower lip
	Orbicularis Oris	Maxilla and mandible	Skin around the lips
	Buccinator	Pterygomandibular ligament and lateral surfaces of mandible and maxilla	Orbicularis oris

Marginal Mandibular	Depressor Anguli Oris (L)	*as above*	
	Depressor labii inferioris (M)	*as above*	
	Mentalis (N)	Mandible	Skin of the chin near midline
Cervical	Platysma	Inferior margin of the body of the mandible	Skin over the upper portion of the breast area